sented that acceleration of fibrinolysis is due to flooding of the circulation by plasminogen activator and that prolongation is probably due to an inhibitor. Prolonged prothrombin time, increased prothrombin consumption index, acceleration of the heparin-retarded clotting time, and fall in platelet count are also frequent during the first hours after injury. There is evidence also of an early deficiency in factor V and a fall in factor VII and prothrombin. The following days are characterized by continued prolongation of fibrinolysis, a lengthening of clotting time, and an increased prothrombin consumption index suggestive of a defect in thromboplastin generation. Prothrombin time may vary phasically. While plasma prothrombin and factor VII are reduced, there is now little change in factor V. The platelet count continues to fall for a few days, but then a thrombocytosis develops. Plasma fibrinogen rises within 24 hours and reaches a maximum within a few days; in the severely injured it remains high for long periods. Various changes are influenced by the severity of trauma.

-- J.Am. Med. Assn. References & Reviews

433 Fatigue, Insufficiency, and Pathologic Fractures. R.L. Pentecost, R.A.Murray, and H.H. Brindley. J.Am.Med. Assn. 187, 1001-1004 (March 28, 1964).

A partial or complete fracture which results from the inherent inability of bone to withstand stress applied without violence in a rhythmical, repeated, sub-threshold manner is a "stress fracture." Because numerous terms have been used in the medical literature to designate the two types of stress fractures, the following classification is recommended. A "fatigue fracture" occurs when abnormal stress is applied to bone with normal elastic resistance. An "insufficiency fracture" is produced by normal or physiological stress applied to bone with deficient elastic resistance. Fatigue and insufficiency fractures occur most frequently in the weight-bearing bones. The term "pathologic fracture" should be limited to any fracture in bone weakened by tumor.

-- Authors' abst.

434 Physiological Studies of Pulmonary Edema at High Altitude. H.N. Hultgren. Circulation 29, 393 (March, 1964).

Cardiac catheterization studies were performed in 4 patients during acute pulmonary edema at an elevation of 12,300 feet in the central Peruvian Andes. Pulmonary hypertension, low cardiac output, arterial unsaturation, and low normal pulmonary artery wedge pressures were observed. Oxygen breathing was accompanied by a prompt, marked fall in pulmonary artery pressure and a slight rise in wedge pressure indicating the presence of anoxic pulmonary arteriolar constriction. In one patient, pulmonary artery wedge pressures were not elevated during added hypoxia nor during exercise. The blood pressure response to the Valsalva maneuver was normal. Similar studies were carried out in 4 subjects after recovery from pulmonary edema. One 9-year-old boy had persisting pulmonary hypertension. None had evidence of underlying cardiac disease.

-- J.Am. Med. Assn. References & Reviews

435 Respiratory Infection Following Tracheostomy. M.S. Gotsman and J.L. Whitby. Thorax 19, 89 (Jan. 1964).

There are many enthusiastic advocates of tracheostomy, since it has been found valuable in the treatment of poliomyelitis, tetanus, and other conditions associated with respiratory paralysis. However, little attention has been paid to the postoperative respiratory sequels. The authors have been impressed by the frequency with which these patients have become infected with organisms causing cross-infections in hospitals, particularly staphylococci. In order to assess the incidence and significance of infections after tracheostomy, they examined 29 patients submitted to tracheostomy in their hospital during 1961, for evidence of pulmonary infection. Twenty-three lived for more than 3 days and 18 of these became infected